CLINICAL NOTES AND CASE REPORTS

PNEUMONIA RECURRING TEN TIMES IN A BOY OF TWELVE YEARS*

REPORT OF A CASE

By HENRY HERBERT, M. D.
Los Angeles

S. S., male, now aged fourteen years, had his first attack of lobar pneumonia in Los Angeles at the age of two years. Thereafter he suffered one attack every year, and even had two attacks during one year in Arizona. All in all he had seven attacks in Los Angeles and three in Arizona. I observed all of the Los Angeles attacks.

He is the oldest of four children. The other three are healthy, as are the parents. The patient has had bronchial asthma since infancy; I have observed it since the first attack of pneumonia.

The attacks run a rather mild course; there are chills, continuous fever, cough, rusty sputum, and bronchial breathing in either of the lower lobes. The disease ends usually by lysis, with complete recovery in two or three weeks, leaving no sequelae. All of the attacks occur in winter, and during the interim the boy is in very good condition. He is of normal build and strength.

Laboratory tests during the last attack in 1926 revealed in the blood a leukocyte count of 21,000, with 83 per cent polymorphonuclears, and in the sputum the usual array of bacilli characteristic of lobar pneumonia. About three weeks after the onset a roentgenogram revealed considerably more than the usual thickening throughout both hilar and perihilar regions, while the periphery and apices of both lungs were clear—the picture produced by repeated acute pulmonary infection.

According to Norris and Landis (textbook), "No other disease is so prone to recur in the same individual and not a few instances are on record of individuals who had ten or more attacks. In the often quoted case of Benjamin Rush no less than twenty-eight attacks occurred."

Chomel (cited from Lord's textbook) observed as many as ten attacks in the same patient. It may be safely stated that, among every one hundred pneumonia cases, almost regularly fifty represent first attacks, thirty-two second, fifteen third, and the remaining three represent fourth and even more frequently repeated attacks.

In the summer of 1927 prophylactic treatment with pneumococcic vaccine was given and for the first time he had no pneumonic attack in that year. The same prophylactic treatment was given in September 1928.

There are two theories advanced regarding the occurrence of such repetition: one, that there is a nidus or a "permanent" focus of infection, which flares up in case of a cold or bronchitis; the case above does not correspond to this theory, because the pneumonia did not appear always in the same lobe or on the same side.

The second theory pertains to lack of immunity or lack of resistance against this specific kind of bacteria; and the subsequent immunization on the pneumococci vaccine seems to favor the latter conception of this rare phenomenon.

In regard to prognosis, it is a fact that the mortality in children with pneumonia is low.

1052 West Sixth Street.

* Read before the Los Angeles County Medical Society, October 4, 1928.

* From the Los Angeles General Hospital.

CALCIUM METABOLISM IN MARBLE BONE (ALBERS-SCHÖNBERG DISEASE)

REPORT OF A CASE

By RANDOLPH G. FLOOD, M. D.
San Francisco

IT is the purpose of this paper to report the calcium metabolism findings in a case of Albers-Schönberg disease rather than a detailed clinical report, as this will be done by other authors in a future communication.

Miss Janet T., age 11 years, was referred to me November 7, 1928, by Dr. Leo Eloesser, with the request that I do a calcium balance on her. The family history was essentially negative. Her past history included measles, mumps, and pertussis. She gave a history of repeated bone fractures, three fractures of the left femur starting at the age of eight years, with slow periods of healing between. In December of 1927 she fractured the right femur, which was put in a cast for two months. She was again allowed up, and fractured the right femur in the same place in March of 1928. Two months later the x-ray showed an imperfect union. Doctor Eloesser was called in and the leg reset, and the diagnosis of marble bone was made, which was confirmed by the x-ray plates. At this time it was observed that she had a visual defect which, on examination, proved, due to a bilateral primary optic atrophy, most advanced in the right eye. This completed the picture of Albers-Schönberg syndrome.

She was admitted to St. Mary's Hospital on November 8, 1928. A complete physical examination was done which showed a markedly undersized girl weighing 21.6 kilos. The only physical abnormalities noted, except for the fractured right femur, was a head with rather pronounced frontal and parietal bosses, moderate pallor and bilateral optic atrophy most marked in the right eye.

Routine laboratory tests were negative except for a moderate anemia: 75 per cent hemoglobin; red blood corpuscles, 4,200,000; and many large calcium oxalate crystals in the urine.

On admission she was placed in a modified metabolism bed and started on a selected diet, the CaO contents of which was determined by wet-ashing an aliquot portion of the entire mixed diet. She was kept on this diet for a period of three days to put her in a calcium equilibrium with her prescribed intake. On the third day a blood calcium determination was made, and on the morning of the fourth day the metabolism period was started. The stool was marked by giving 0.5 gram carmin by mouth. All stools were collected after the carmin appeared for a period of three days.

The urine was marked by the injection of one cubic centimeter phenolphthalein, and all urine collected after the appearance of the phthalein.

All food was carefully weighed during the next three days and CaO determination made on aliquot parts from which the total CaO intake was estimated.

All stools and urine were collected for the next seventy-two hours and at the end of this time were again marked by the administration of carmin and phthalein which, when they appeared, ended the experiment.

All CaO determinations were made by the McCrudden method, checking with volumetric and with gravimetric determination when possible.

Throughout the experiment, daily blood CaO determinations were made and all results charted under headings, November 29, 30; December 1, 2, and 3.

On examining the chart the first thing that draws attention is the persistent hypercalcium present, which is of a high degree, ranging from 15.8 to 16.6 milligrams of CaO, whereas the normal range is from 8.8 to 10 milligrams per 100 cubic centimeters of blood.

DATE	Nov. 29.	Nov. 30	Dec 1	Dec 2	Dec 3	Jan 8	Jan 11	Jan 13	Jan 16	Jan 17	Jan 18	Jan 19
TOTAL CaO INTAKE		5.1665 gm.									4.8862	
TOTAL URINE OUTPUT		2440. cc				1210	975	1410	1160	3710.		
CaO in 200 c.c. URINE		0.0053				.0051	.0044	.0026	.0044		.0037	
CaO in TOTAL URINE		0.0649				.0310	.0213	.0198	.0256		.0684	
WT. OF STOOL		35.3				GIVEN 10 UNITS PARATHYROID	GIVEN 15 UNITS	GIVEN 20 UNITS	GIVEN 25 UNITS (VOMITED)		41.27	
CaO in 5 gms stool		0.4806									0.5969	
CaO in TOTAL STOOL		3.3918									4.9276	
TOTAL CaO-OUTPUT-3 DAYS		3.4567									4.9960	
TOTAL CaO-RETENTION		+ 1.7098									− 0.1098	
CaO-RETENTION-PER-KILO-3 DAYS		+ 0.0803								−		
CaO-RETENTION-PER-KILO-PER-DAY		+ 0.0288								−		
BLOOD CaO Mgs.	16.4	16.4	15.7	16.6	15.8	14.6	14.6	15.0	15.2	15.2	15.0	15.3
24 hr. URINE-CaO-OUTPUT Mgs.			0.0218			0.0310	0.0213	.0198	.0256		.0228	
BODY WT. KILOS.		21.6								24.2		

The second interesting point is that even though the child has a high blood calcium her calcium retention is within normal limits: 28.8 milligrams per kilogram body weight per day. The third and most striking observation is that, with this marked hypercalcemia, the CaO output in the urine was markedly under the normal, being only 0.0218 milligrams per day. Our observations, as well as those of L. A. Hoag et al. (*American Journal Diseases of Children*, 33, 910-925, June 1927), are that the human compensates for increased blood calcium produced by parathyroid injections by excreting the CaO in the urine, this being contrary to the dog, who compensates through increased fecal output.

This last observation seemed to indicate that this child's renal threshold for calcium was evidently raised. We decided to bring her into the hospital again, raise the blood calcium by injections of parathyroid, and find the height to which the blood CaO would have to be elevated to overstep this threshold.

With this in view we brought her in on January 8, 1929, and made a preliminary blood CaO determination. To our surprise we found a hypercalcemia of only 14.6 milligrams per 100 cubic centimeters instead of 15.8, our last determination. This finding was carefully checked and found correct. This observation of fluctuating blood calcium in marble bone has previously been described as the "calcium tide" by M. B. Schmidt (Kalkmelastase und Kallsgicht, *Deutsche med. Wchnschr.*, 1913, No. 2). Evidently at this time we found her on the low ebb of her tide.

We then gave her 10 units of Collips' parathyroid intramuscularly, with one cubic centimeter phthalein to mark the urine. All urine was collected for the next twenty-four hours, and again marked with phthalein and the CaO contents estimated in the twenty-four-hour output. Twelve hours after the injection, when the maximum effect of the parathyroid was supposed to be reached, a blood CaO determination was made, as we wished to see how high the blood calcium would have to rise before an appreciable increase in CaO appeared in the urine.

Following the 10 units there was no increase in blood calcium or the calcium output in the urine.

We repeated the experiment three days later, using 15 units of parathyroid, also without results. Again three days later 20 units were given; this raised the blood calcium to 15 milligrams, but caused no increase in the urinary calcium.

Three days later we gave 25 units with a definite increase of blood calcium, but still no increase in urine calcium. At this point the child began to vomit and have epigastric distress, and we were afraid to increase the dose of parathyroid.

Evidently the kidneys are impervious to a sudden increase of 5 per cent of blood calcium.

Another interesting observation is the fact that the child became upset and reacted when the blood calcium was suddenly increased 5 per cent, but was still under one milligram per 100 cubic centimeters of the maximum existing at the height of her tide during the metabolism period.

The explanation is that she can adjust herself to gradual changes in blood calcium within wide ranges, provided the transition is not too rapid.

Having found that we could not increase the CaO output in the urine by suddenly increasing the blood calcium, we decided to again run a calcium balance while she was held at this high artificial level, with 25 units of parathyroid given at thirty-six-hour intervals.

We repeated the same technique as in our original metabolism period and charted the findings under January 17, 18, and 19.

Examination of these figures show that we produced a slight negative balance, the child losing 0.1098 grams CaO daily.

However, we feel that this is only a temporary small negative balance, as the child will readjust herself to this blood calcium level with a positive CaO retention within a short time, as she did when she was at the height of her tide in the first experiment with a blood calcium of 16.6 milligrams, which is considerably higher than her present hypercalcemia of 15.3.

CONCLUSIONS

Evidently, then, this child has a kidney whose high threshold is fixed and, to all intents, impervious to increased blood calcium.

The child is able to adapt herself to wide fluctuations in blood calcium, provided the transition is gradual, but reacts to sudden change, much as a normal person.

There is little hope in giving this child a negative calcium balance by the administration of Collips' parathyroid, as she has the ability of gradually adjusting herself to marked hypercalcemia with a definite CaO retention.

490 Post Street.

CARCINOMA OF BREAST WITH DIFFUSE CARCINOMA OF STOMACH

REPORT OF A CASE

By Henry S. Penn, M. D.
Boston, Mass.

THE case here reported is one of more than unusual interest from the standpoint of the pathology involved and the age of the patient.

Mrs. O. O., age 26, was referred to me in November 1926 with the following history:

Chief Complaint.—Pain in the region of the right breast.

Family History.—Father, mother, one brother and one sister living and well.

Present Illness.—Patient was well until about six months ago, when she first noticed a small lump in the right breast. The growth has since increased in size and the patient has lately suffered sharp pains over the right pectoral region and axilla. She has lost about ten pounds within the last three months. Her appetite has been fairly good, but she seems to be losing strength.